symptoms than they would have had if the PDA had remained open.

Another important aspect is the accurate measurement of pulmonary vascular resistance.[9] For patients breathing oxygen, it is crucial to include values for dissolved oxygen in the calculation of resistance. Failure to include dissolved oxygen in the measurement of pulmonary blood flow results in an underestimation of resistance. Many cardiac catheterisation laboratories do not have the facilities for the measurement of oxygen consumption,[9] and instead, assumed values from tables are used. This inevitably provides an additional potential source of inaccuracy in pulmonary vascular resistance measurements. It is not clear from the text if assumed oxygen consumption was used or if resistance measurements took dissolved oxygen into account.

So, while on the one hand, it is possible to safely close a very large arterial duct in patients with pulmonary hypertension, it seems inevitable that at least some will develop progressive and fatal pulmonary vascular disease and eventually be worse-off than they would have been in terms of symptoms and life expectancy. Some patients had resistance values >15 units. The key to the safe management of these patients is the accurate measurement of pulmonary vascular resistance and management based on a resistance value rather than direct measurement of pulmonary artery pressure, which is never an accurate reflection of resistance. Management based on the direct measurement of pulmonary artery pressure during duct occlusion, although in common practice, is inevitably flawed.

Finally, in patients such as these, it is arguable that successful duct closure should be followed by an aggressive therapeutic approach to reduce pulmonary vascular resistance using pulmonary vasodilator agents, if possible.

Such treatment is of course extremely expensive, rarely successful in achieving a normal resistance and inappropriate in many developing countries, and should be part of research protocols and audit. For the group of patients reported, it is important that follow-up should include measurement of pulmonary vascular resistance at a given period after closure of the duct. Such a follow-up study might well be worthy of publication and will help to enhance our understanding of pulmonary vascular disease in this group of patients. However, the management of patients with a raised pulmonary vascular resistance and patent arterial duct must always be based on sound and reproducible physiological measurements and the underlying principle of ''first do no harm.''

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